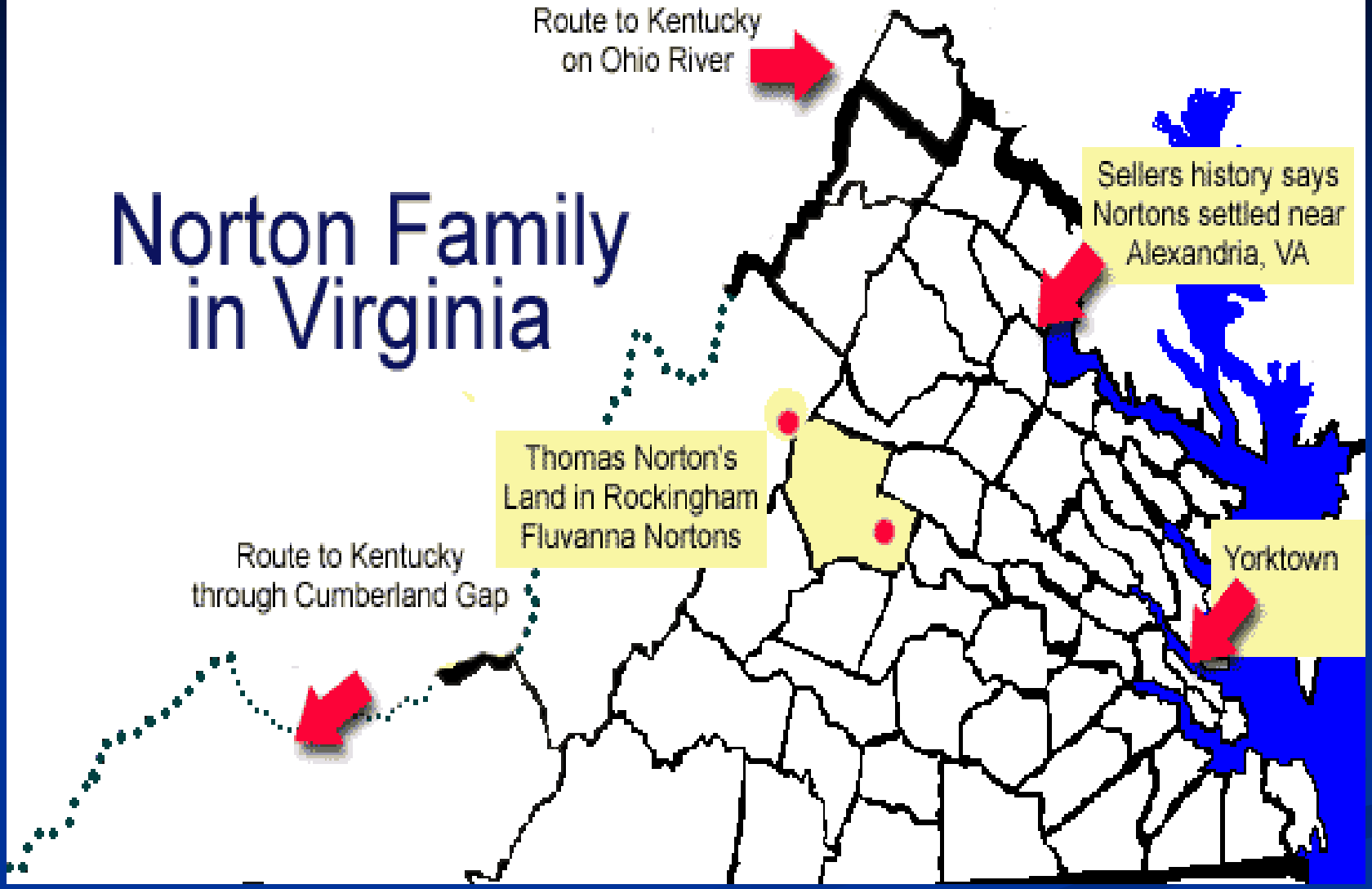
Chronic Obstructive Pulmonary Disease

Maj David Norton, USAF, MC
Pulmonary/Critical Care Medicine
Malcolm Grow Medical Center
Andrews AFB, MD

Norton Family in Virginia



Plan of Attack

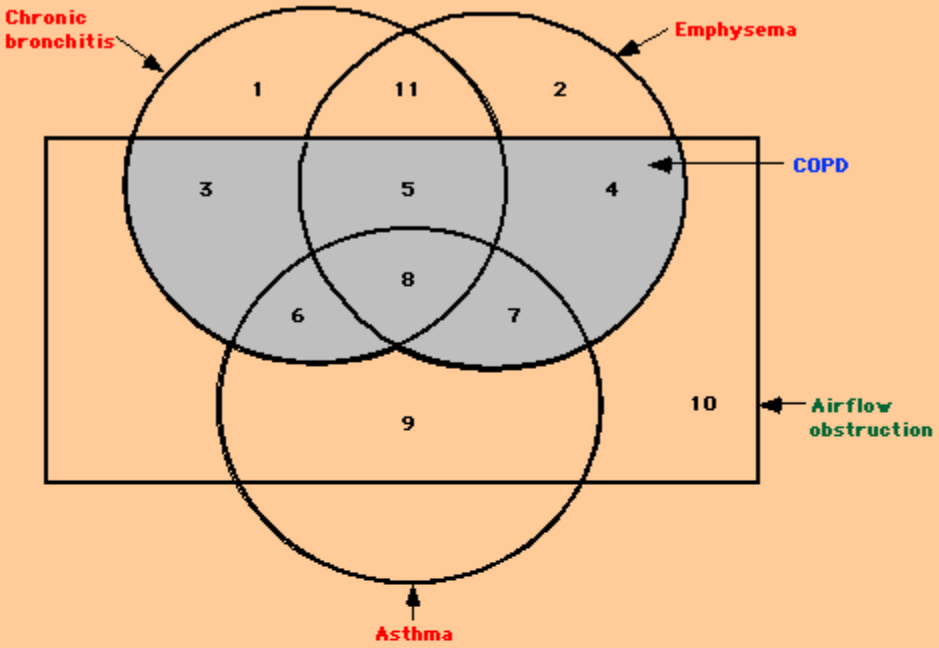
- Definitions
- Epidemiology
- Diagnosis
- Managing Stable COPD
- Managing Acute Exacerbations of COPD

Definitions

- “A disease state characterized by airflow limitation that is not fully reversible. Airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. Symptoms, functional abnormalities, and complications of COPD can all be explained on the basis of this underlying inflammation and the resulting pathology.”
- Global initiative for chronic obstructive pulmonary disease

Definitions

- Chronic Bronchitis (clinical)
  - Sputum production more days than not for at least 3 months a year for at least 2 years
- Emphysema (pathologic)
  - Parenchymal destruction airspace walls distal to terminal bronchioles, without fibrosis
- Important: You can have either, but to have COPD you MUST demonstrate obstruction (thus the “O” in COPD)



Chronic obstructive pulmonary disease This nonproportional Venn diagram shows subsets of patients with chronic bronchitis, emphysema, and asthma (black circles). The subsets defined as COPD are shaded gray. Subset areas are not proportional to actual relative subset sizes. Asthma is, by definition, associated with reversible airflow obstruction; in variant asthma special maneuvers may be necessary to make the obstruction evident. Patients with asthma whose airflow obstruction is completely reversible (subset 9) are not considered to have COPD. In many cases it is virtually impossible to differentiate patients with asthma whose airflow obstruction does not remit completely from persons with chronic bronchitis and emphysema who have partially reversible airflow obstruction with airway hyperreactivity. Thus, patients with unremitting asthma are classified as having COPD (subsets 6, 7 and 8). Chronic bronchitis and emphysema with airflow obstruction usually occur together (subset 5), and some patients may have asthma associated with these two disorders (subset 8). Individuals with asthma exposed to chronic irritation, as from cigarette smoke, may develop chronic productive cough, a feature of chronic bronchitis (subset 6). Such patients are often referred to in the United States as having asthmatic bronchitis or the asthmatic form of COPD. Persons with chronic bronchitis or emphysema without airflow obstruction (subsets 1,2 and 11) are not classified as having COPD. Patients with airway obstruction due to diseases with known etiology or specific pathology, such as cystic fibrosis or obliterative bronchiolitis (subset 10), are not included in this definition.

Epidemiology

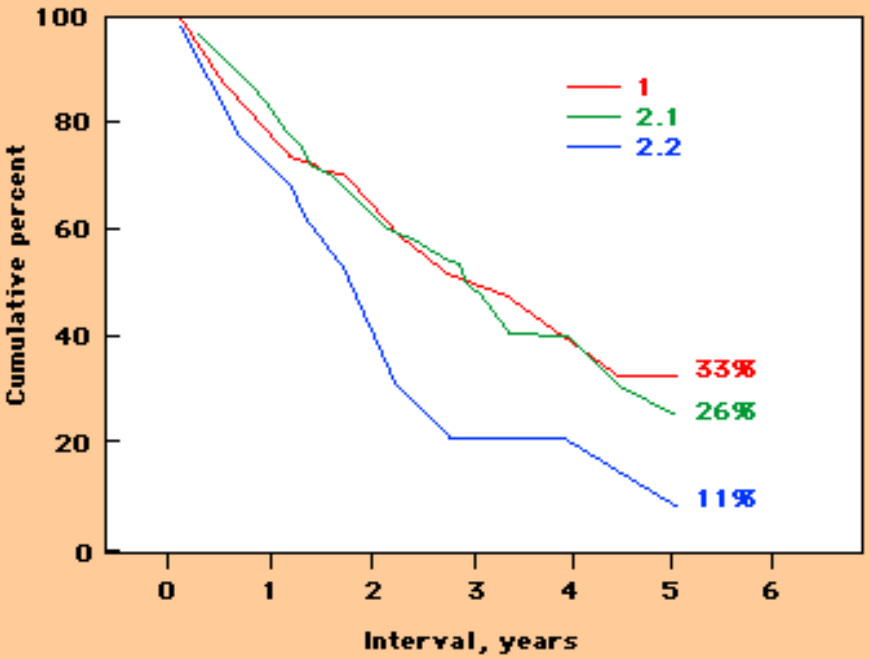
- Fourth leading cause of death in U.S.
- 100,000 American deaths each year
- 15-20% of chronic smokers develop COPD
- 2.5% mortality for COPD hospital admissions
- COPD with acute respiratory failure:
  - 24% in hospital mortality
  - 59% one year mortality

Epidemiology

Survival Rates in COPD According to Percent Predicted Postbronchodilator FEV1 (PB FEV1) in Patients ≤ 65 Years of Age

Initial PB FEV1 (% predicted)	Number	Cumulative survival rate, percent			
		At 2 years	At 5 years	At 10 years	At 15 years
<20	9	44	11	11	0
20-29	40	65	30	10	3
30-39	43	83	47	21	7
40-49	26	92	89	39	30
50-59	21	95	95	57	32
60+	9	100	89	89	67

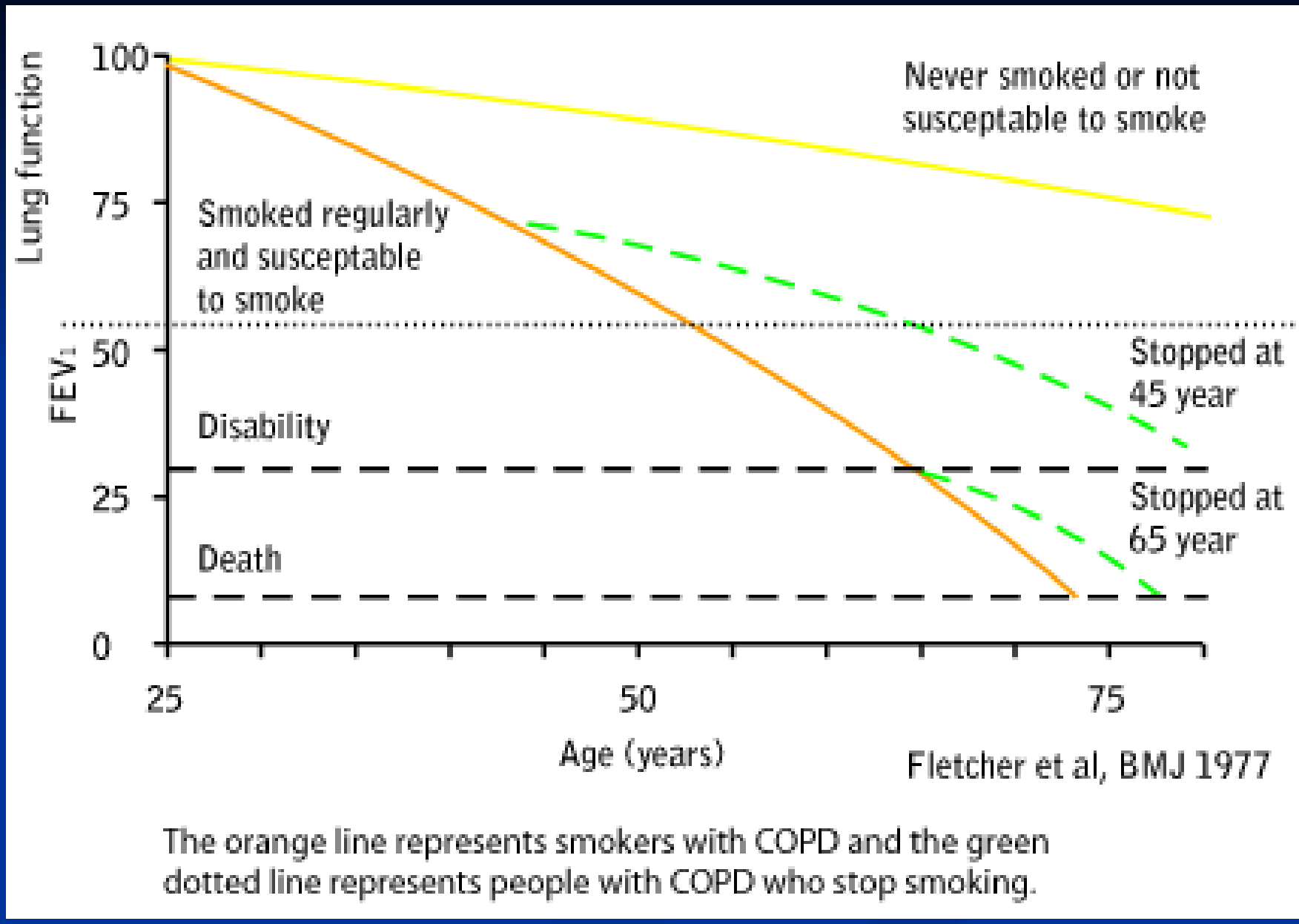
Epidemiology



Survival in COPD Kaplan-Meier survival curves for nonhypercapnic patients (type 1), reversible hypercapnics (type 2.1), and chronic hypercapnic patients (type 2.2). Survival among type 2.2 patients was significantly worse ($P<0.05$) by the Cox-Mantel test) than the other two groups. Percentages refer to proportion of patients who survived 5 years. (Redrawn from Costello, R, Deegan, P, Fitzpatrick, M, McNicholas, WT, Am J Med 1997; 103:239.)

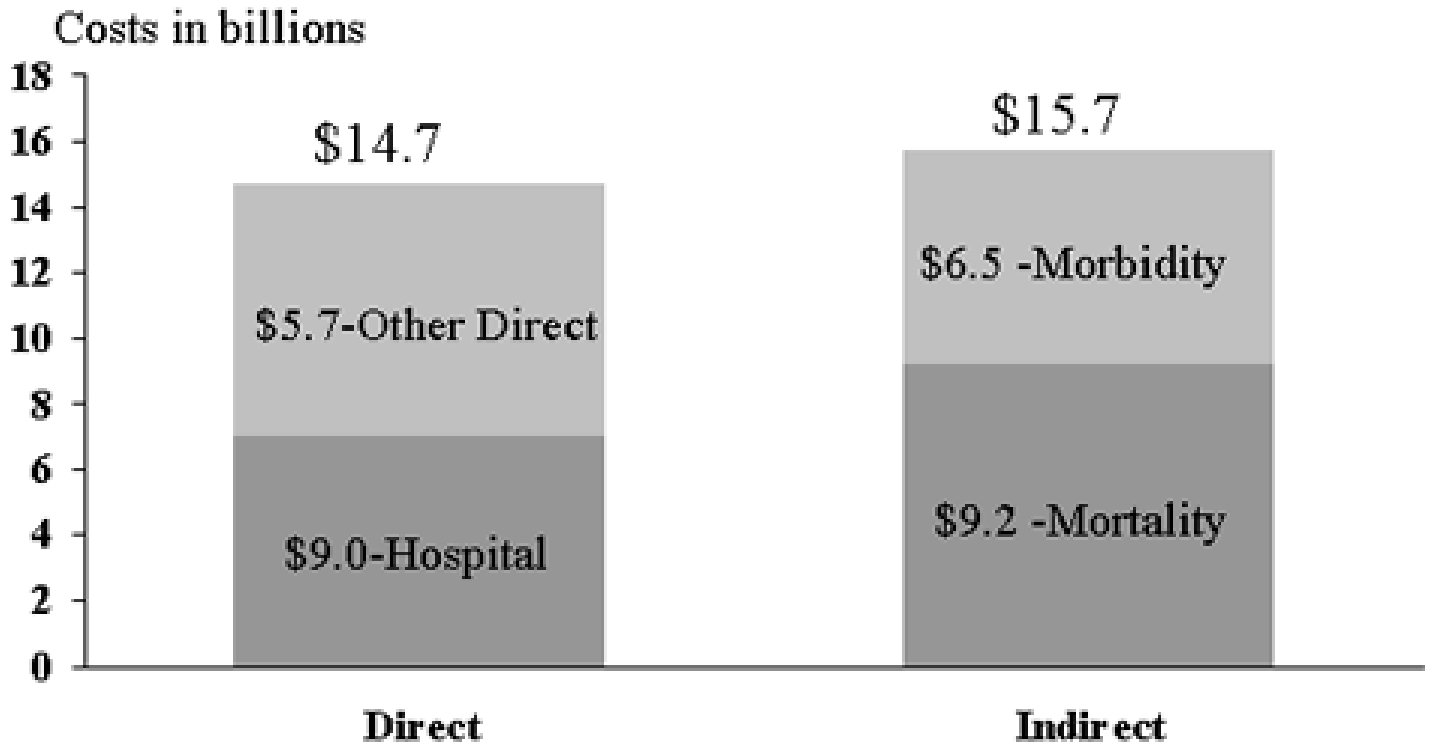
Epidemiology

- If you have COPD and $PaCO_2$ > 50mmHg:
  - 67% chance of being alive in 6 months
  - 57% chance of being alive in 12 months
- Bad monkey! Those green bananas aren’t for you.



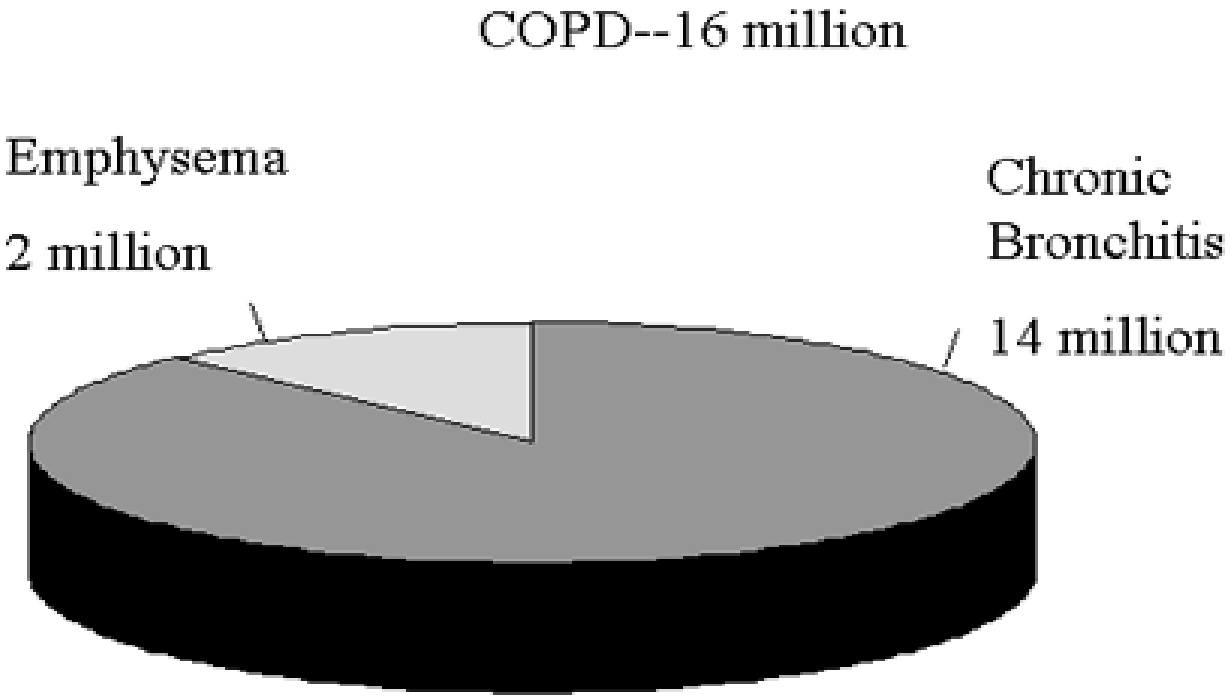
The orange line represents smokers with COPD and the green dotted line represents people with COPD who stop smoking.

Figure 7.
Estimated cost of COPD, 2000



Source: *Morbidity and Mortality: 2000 Chartbook on Cardiovascular, Lung, and Blood Diseases*, National Heart, Lung, and Blood Institute

Figure 1. Prevalence of Chronic Obstructive Pulmonary Disease, U.S., 1996



Source: National Center for Health Statistics, CDC, 1996

THE WHITE HOUSE

WASHINGTON

October 24, 2002

I send greetings to all those participating in the United States Chronic Obstructive Pulmonary Disease Coalition.

Chronic Obstructive Pulmonary Disease (COPD) is the fourth leading cause of death in our Nation. Approximately 16 million Americans suffer from this progressive disease of the lungs that is associated mainly with chronic bronchitis and emphysema. Every year an estimated 100,000 individuals die from COPD.

To improve the quality of life of all our citizens, my Administration is dedicated to increased Federal funding for medical research at the National Institutes of Health (NIH). Through the National Heart, Lung, and Blood Institute at the NIH, we are supporting research to better understand the causes of COPD, and to improve prevention and treatment. I have also introduced my HealthierUS initiative to help Americans avoid chronic conditions, such as COPD, through modest improvements in fitness and lifestyle.

I commend the organizations of the United States Chronic Obstructive Pulmonary Disease Coalition for your efforts to increase awareness of COPD, and I applaud your work to advance care for those affected by this disease. By working together, we bring hope to countless citizens, and create a brighter future for all Americans.

Laura joins me in sending our best wishes.

Diagnosis

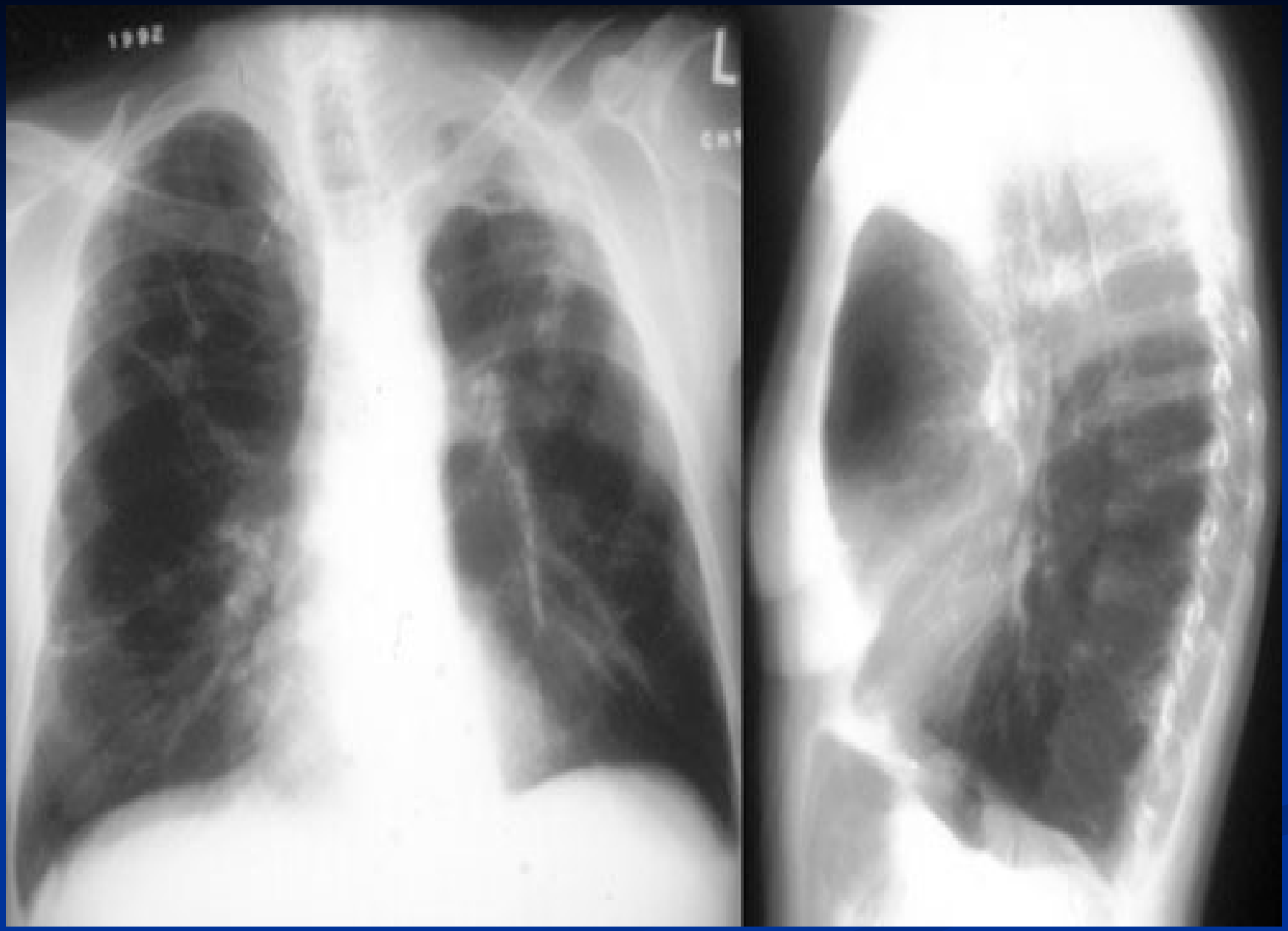
- Symptoms
  - Dyspnea
  - Sputum production (especially in the morning)
  - Recurrent acute chest illnesses
  - Headache in the morning – possible hypercapnia
  - Cor pulmonale (R heart failure)

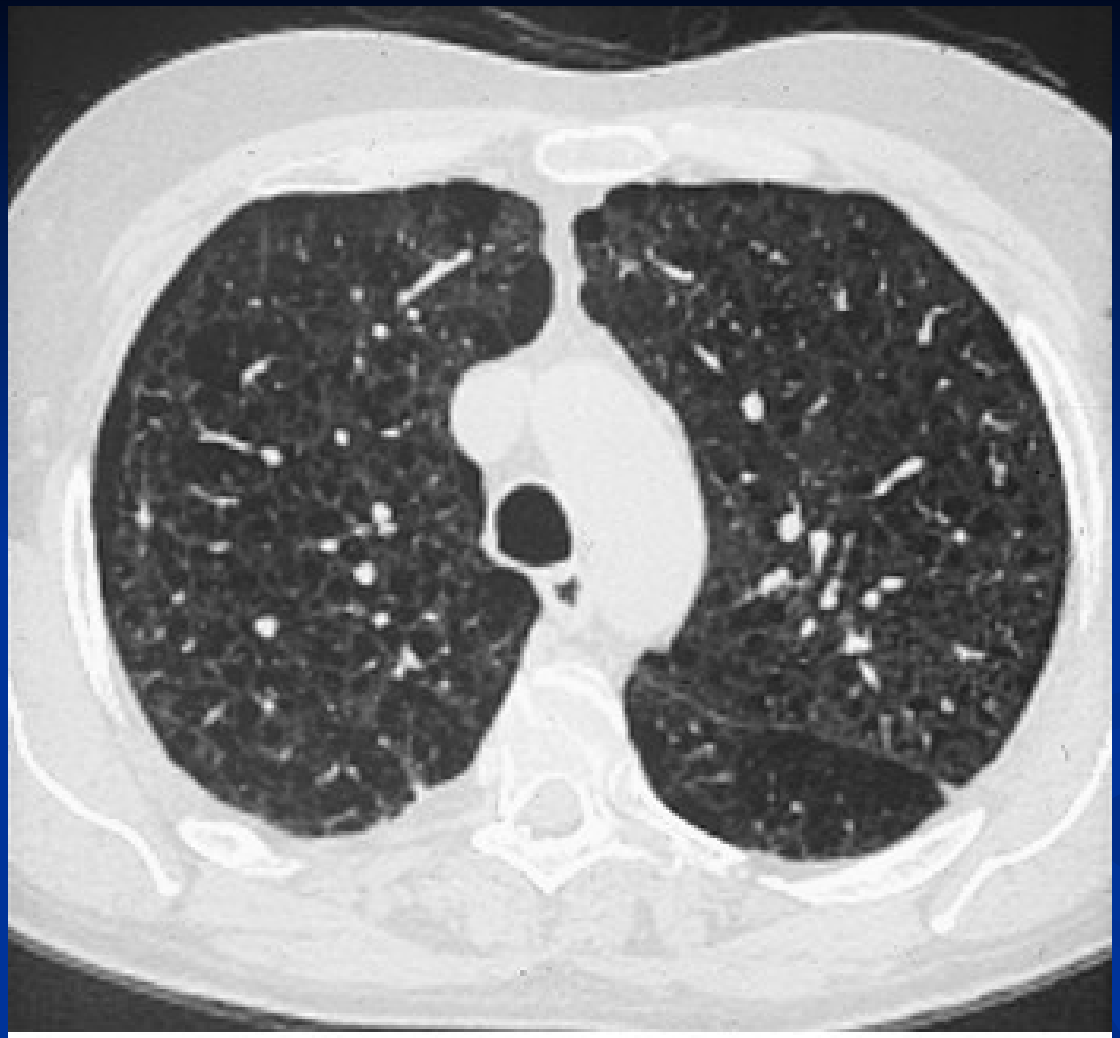
Diagnosis

- Signs
  - Prolonged expiratory time
  - Expiratory wheezes
  - Increased AP diameter of chest
  - Decreased breath sounds (especially upper lung fields)
  - Distant heart sounds
  - End stage: accessory muscles, pursed lip breathing, cyanosis, enlarged liver

Diagnosis

- Radiology
- Chest X-ray
  - Bullae, often bilateral upper lobes in smokers
  - Flat diaphragms (best seen on lateral) and retrosternal airspace can indicate air trapping
- High Resolution CT of Chest
  - Most sensitive to detect above changes
  - No role in routine care of COPD patients
  - Can be useful for giant bullous disease surgeries or lung volume reduction surgery planning





Centrilobular emphysema HRCT shows multiple small lucencies permeating the upper lobes. The wall of the emphysematous spaces is imperceptible. Courtesy of Paul Stark, MD.

Diagnosis

- Pulmonary Function Testing

- Spirometry: Decreased FEV1/FVC
  - FEV1 percent predicted defines severity
- Lung volumes: Increased TLC, RV, RV/TLC
- DLCO: Decreased

Diagnosis

- GOLD Staging Criteria
- Stage O: Normal spirometry; chronic sx
- Stage 1 (Mild):
  - FEV1/FVC < 70%; FEV1 > 80% predicted
- Stage 2 (Moderate):
  - FEV1/FVC < 70%; FEV1 30-80% predicted
  - 2A: FEV1 50-80% predicted
  - 2B: FEV1 30-50% predicted

Diagnosis

- Stage 3 (severe):
  - FEV1/FVC < 70% AND:
  - FEV1 < 30% predicted OR:
  - FEV1 < 50% predicted and clinical evidence of R heart failure

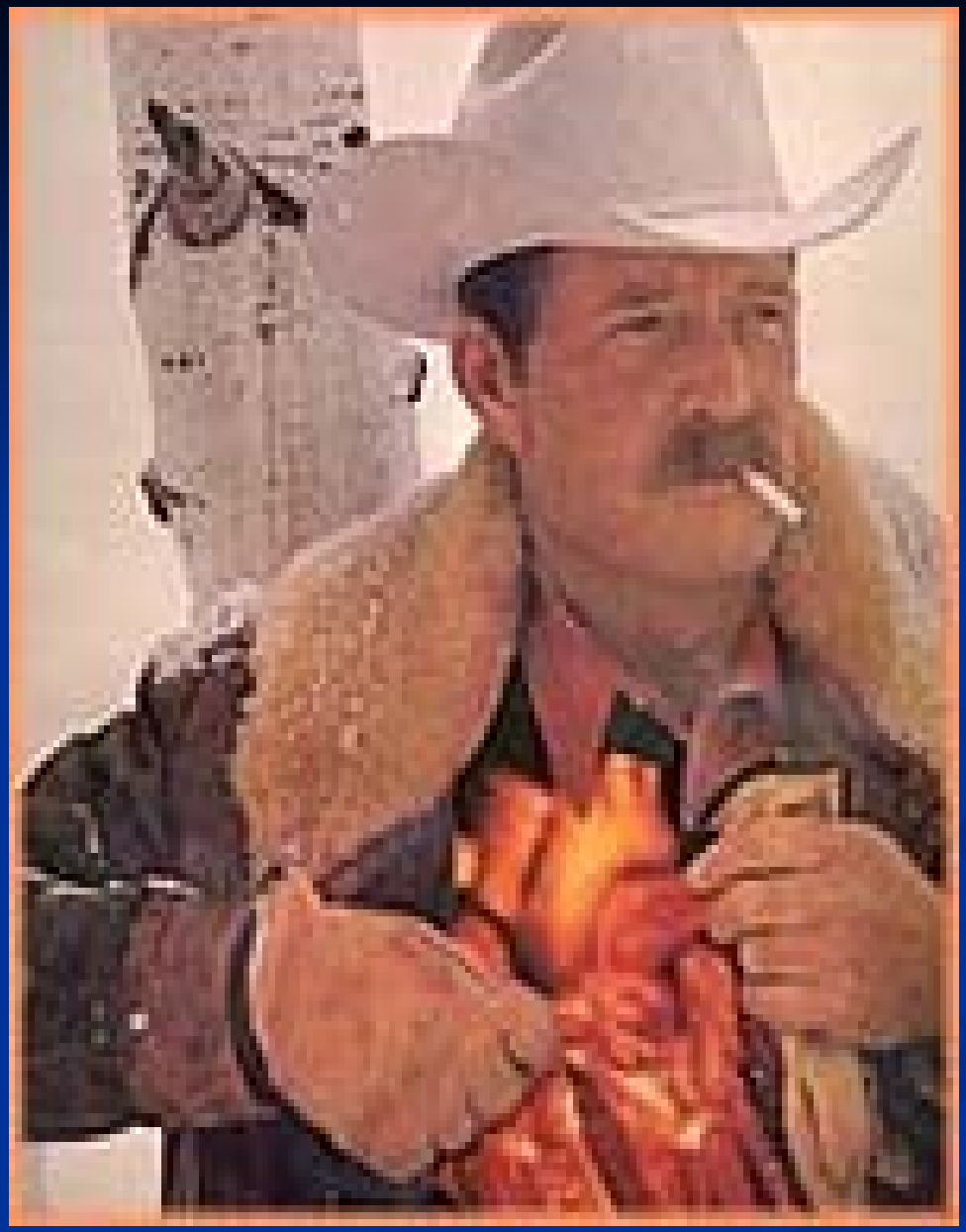
Diagnosis

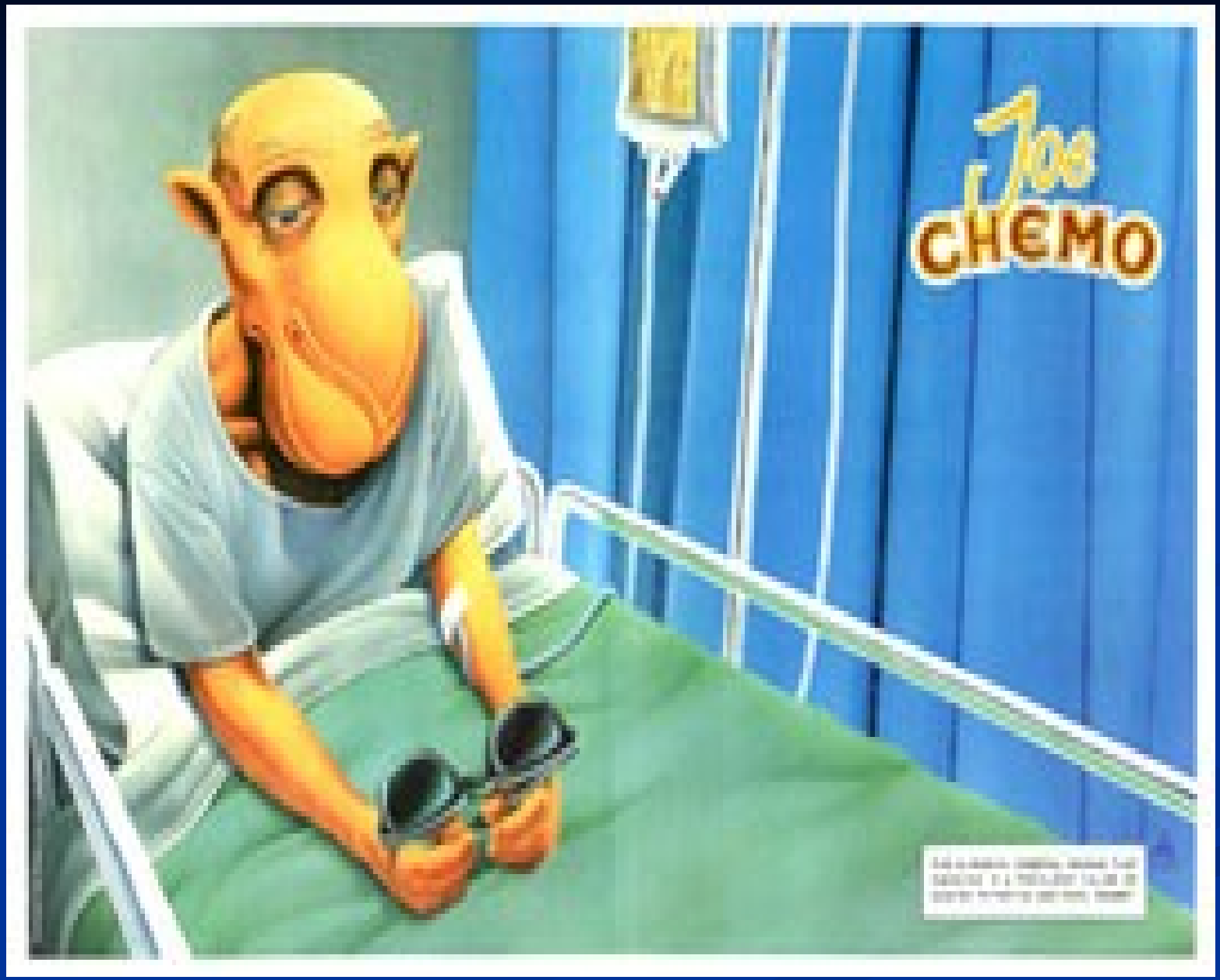
- American Thoracic Society – Spirometry
- Low FEV1/FVC defines obstruction
- FEV1%predicted Category
- < 35% Very Severe
- 35-50% Severe
- 50-60% Moderately Severe
- 60-70% Moderate
- 70-80% Mild
- 80-100% Mild vs. Normal variant
- > 100% Normal

Managing Stable COPD

- Smoking Cessation Is KEY!
  - YOUR intervention will make a difference – must address at each visit
  - Medication, accupuncture, hypnotherapy
- Two therapies ONLY have been shown to improve mortality in stable COPD:
  - 1) Smoking Cessation
  - 2) Oxygen Therapy


Marlboro

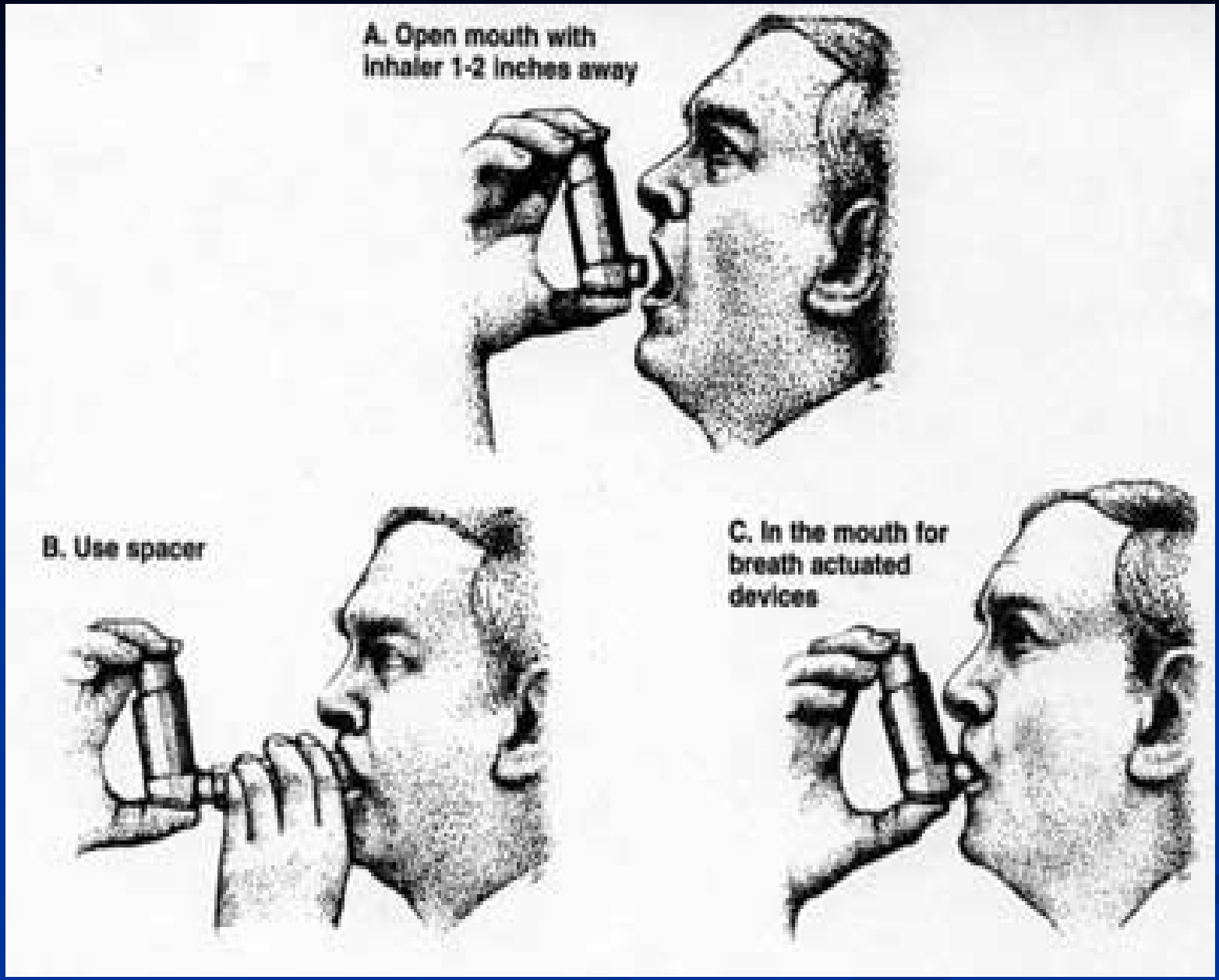



CHEMO

STOP SMOKING IN ½ HOUR FLAT

Managing Stable COPD

- Bronchodilator Technique
  - MDI's get better drug deposition than nebs
    - Use a spacer device with MDI's
  - Technique is key – impt for patient and MD
  - Inadequate dosing can hamper treatment


A. Open mouth with inhaler 1-2 inches away
B. Use spacer
C. In the mouth for breath actuated devices

Managing Stable COPD

- Sympathomimetics
  - Beta-2 selectivity is good
  - Unclear if prn vs. scheduled is better
  - Some additive vs. slightly synergistic effects of combining beta-2 agonist and ipratropium (Combivent)
  - Some data to support decreased H.influenzae pneumonia incidence with Serevent

Managing Stable COPD

- Anticholinergic Agents (Atrovent, etc)
  - Similar ability to bronchodilate (in appropriate doses) as beta-agonists
  - Also reduces sputum volume; no change in viscosity
  - Usually under dosed
  - Recommend 4-6 puffs qid

Managing Stable COPD

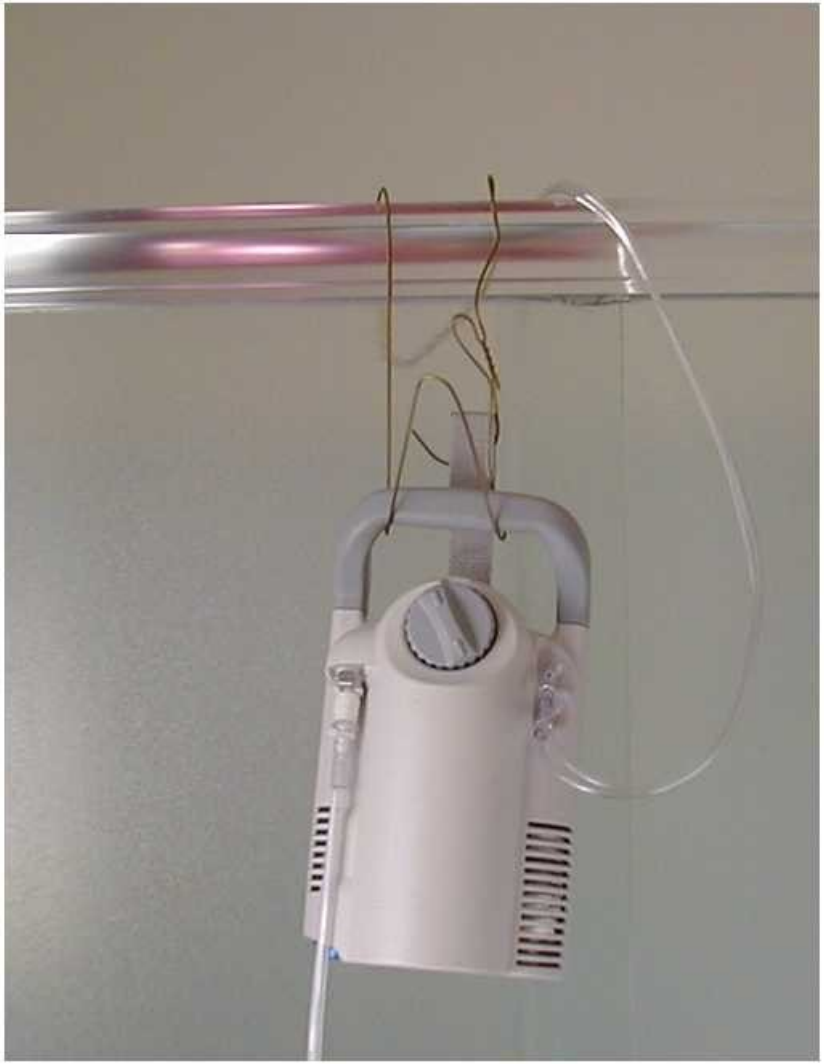
- Theophylline – Be careful
  - Data supporting use are scant, but some improvement in resp muscle function, ABG's – only very modest
  - Significant side effect profile
  - If using, target a serum level of 8-12 mcg/mL
  - RARELY of significant clinical benefit

Managing Stable COPD

- Mucokinetic agents
  - Of no significant clinical benefit in large studies
  - Increased fluid intake DOES NOT affect sputum viscosity significantly
  - Postural drainage and chest PT are generally not useful unless there is a significant bronchiectasis component

Managing Stable COPD

- Oxygen. Yes.
  - Demonstrated to improve exercise performance, symptom indices and mortality
  - Goal in hypercapnic patients for SpO2 need not be greater than 88-90%
  - Always test COPD patients for oxygenation with ambulation if baseline at rest room air SpO2 okay



Managing Stable COPD

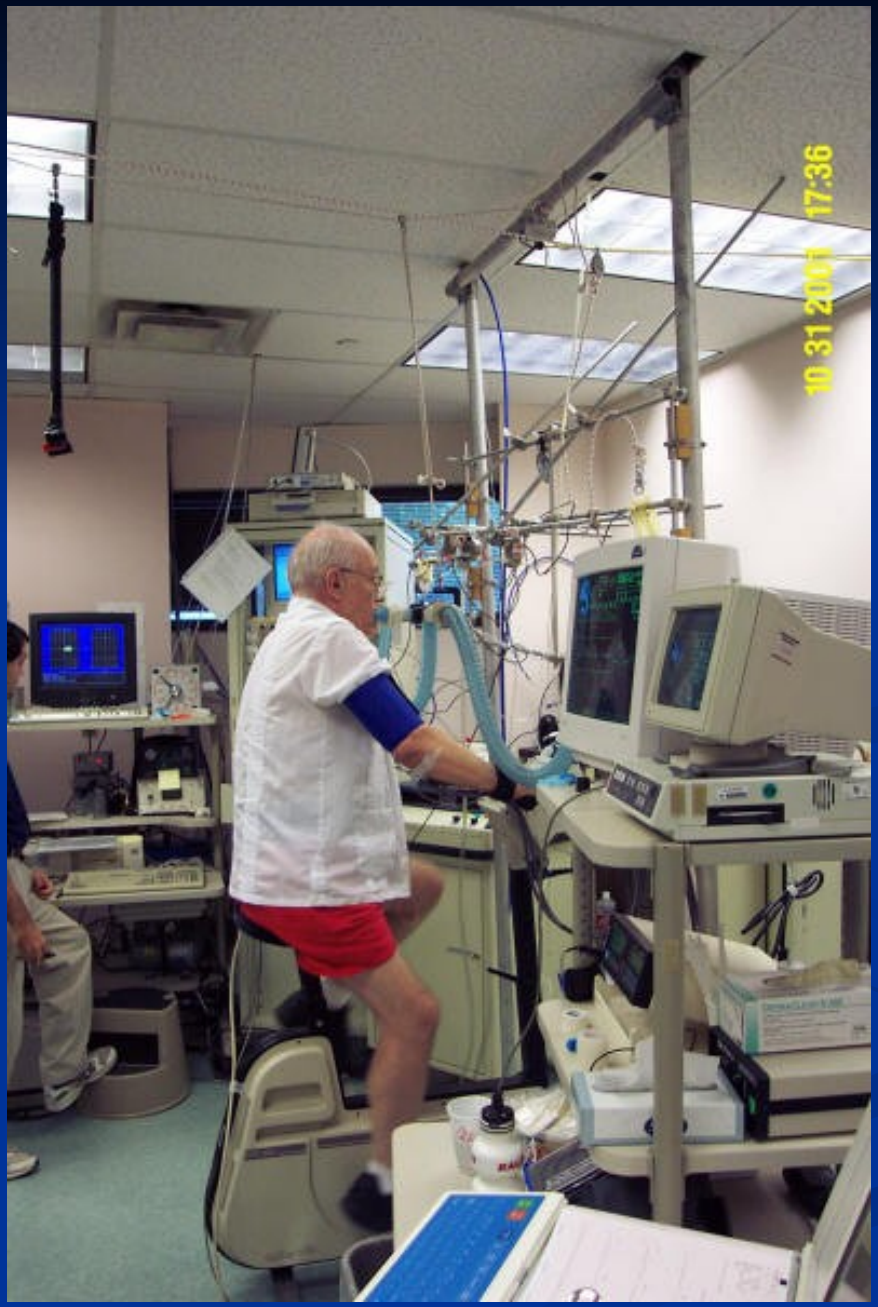
- Systemic Corticosteroids
  - Never demonstrated to significantly impact mortality or exercise capacity
  - Slight improvements in symptom indices
  - Significant side effects
  - Rarely of benefit, generally of harm to your patient
  - Occasionally useful in a small subset failing other therapies AND with demonstrated bronchodilator response on PFT’s

Managing Stable COPD

- Inhaled Corticosteroids
  - Jury still out
  - Lots of recent research with some favorable data supporting its use
  - May be part of standard regimens in the future

Managing Stable COPD

- Vaccines
  - Pneumovax, annual flu shots
- Chronic antibiotic therapy – BAD IDEA
- Nutritional status – Important
- Pulmonary Rehabilitation
  - Improved exercise capacity, symptom scores
- Lung Volume Reduction Surgery
- Transplant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Managing Acute Exacerbations of COPD

- Common precipitants:
  - Infection – esp viral or bacterial
  - Acute bronchospasm
  - Sedation

Managing Acute Exacerbations of COPD

- Who To Admit?
  - Countless studies, few definite answers
  - Worsening hypoxemia and/or hypercapnia
  - Otherwise, mostly a clinical decision (thus your BIG residency salaries!)
- Key points to consider:
  - Oxygen
  - Bronchodilators
  - Steroids
  - Antibiotics

Managing Acute Exacerbations of COPD

- Albuterol:
  - Neb or MDI – neb MAY be better in acute setting, but MDI's have better drug deposition overall
  - Continuous nebulizer treatments confer no benefit over treatments every 1-2 hours
  - Generally should avoid subcutaneous beta-agonists
  - BEWARE: Hypokalemia, tachycardia (occasional)
  - Levalbuterol still with weak clinical data – few situations where it is clinically indicated

Managing Acute Exacerbations of COPD

- ATROVENT (anticholinergic bronchodilator)
  - Bronchodilation
  - May decrease secretions
  - Few significant side effects
  - Usually significantly under dosed – emerging data supports much higher doses than usually used currently

Managing Acute Exacerbations of COPD

- Systemic Corticosteroids
  - Optimal regimen unclear
  - Largest prospective study with benefit used:
    - Solumedrol IV x 3d, followed by prednisone taper over a total of 2 weeks
  - Also frequently used: Prednisone 50qd x 5d (no taper)
  - If good absorption, NO benefit of IV steroids over oral
  - No mortality benefit

Managing Acute Exacerbations of COPD

- Antibiotics
  - “Winnipeg” Criteria (give for 2-3 of the following):
    - Increased cough
    - Increased purulence
    - Increased sputum production
  - Amoxicillin, Doxycycline, TMP/SMX, Azithromycin, Clarithromycin, Levaquin, Tequin all acceptable
  - Details of coverages, optimal abx for a later time…

Managing Acute Exacerbations of COPD

- Mucokinetic Agents – JUST SAY NO.
  - N-acetylcysteine is actually contraindicated in patients with airway obstruction
  - No significant clinical benefit ever demonstrated
  - Chest PT, intermittent positive pressure breathing and postural drainage may actually be harmful in the setting of acute obstruction

Managing Acute Exacerbations of COPD

- Methylxanthines (Theophylline, Aminophylline)
  - Not recommended for acute exacerbations
  - No significant benefit ever demonstrated in large, prospective trials

Managing Acute Exacerbations of COPD

- Oxygen: YES!
  - Generally a good thing – cells like that stuff
  - If requiring a significant increase in FiO2 over baseline requirement, start hunting for something other than just COPD exacerbation
  - BEWARE of CO2 RETAINERS! (goal SpO2 88-90%)
    - 1) Altered V/Q relationships
    - 2) Haldane effect (Hgb*O2 holds less CO2 – goes out into plasma)
    - 3) Decreased ventilatory drive (least impt mechanism)

Managing Acute Exacerbations of COPD

- Non-Invasive Positive Pressure Ventilation
- BiPAP!
  - Set FiO2, inspiratory (IPAP) and expiratory (EPAP)
  - Difference between IPAP and EPAP augments tidal volume, therefore improving minute ventilation. CO2 then gets blown off
  - MORTALITY BENEFIT in patients who will tolerate

Managing Acute Exacerbations of COPD

- Mechanical Ventilation
  - Respiratory distress
  - Acidemia that does not correct quickly with therapy
  - Inability to oxygenate adequately
  - Often a clinical decision relative to patient's work of breathing